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**In the Specification:**

Please insert the following paragraph on page 1 after the title of the invention before line 5:

**CROSS-REFERENCE TO RELATED APPLICATIONS**

The present application is a continuation of U.S. Application No. 09/540,967, filed March 31, 2000, now abandoned.

Please replace the paragraph on page 9, lines 14-17 with the following paragraph:

Another aspect of the invention includes hybridomas which produce monoclonal antibodies of the present invention. One such hybridoma producing rat anti-murine VE-cadherin E4B9 has been deposited with the ATCC, Rockville, Maryland-American Culture Collection (10801 University Blvd., Manassas, VA, 20110-2209 USA (ATCC) on March 31, 2000, and has been assigned accession number PTA-1618.

Please replace the paragraph on page 20, lines 1-9, with the following paragraph:  
Monoclonal Antibody Preparation: Lewis rats (6-8 week old females) were injected subcutaneously (s.c.) with 0.1 ml of protein or peptide [protein concentration??] mixed in Freund's complete adjuvant using a 25-gauge needle. Rats were boosted every 2-3 weeks with antigen and bled via the tail vein every week. After 3 booster immunizations or when sera titers reach maximal levels, mice were sacrificed by CO<sub>2</sub> inhalation. Spleens were recovered from sacrificed animals for monoclonal antibody generation by conventional techniques.

Please replace the paragraph on page 23, lines 15-25 with the following paragraph:

The 20 candidate VE-cadherin antibodies were tested in the "calcium-switch" and "permeability" assays to examine their new junction formation inhibiting activity and existing junction disrupting activity, respectively. Among these 20 antibodies, E4B9 was shown to specifically inhibit adherens junction formation without adversely affecting normal vasculature (FIGS. 3 and 4). Furthermore, the E4B9 antibody was also tested in an *in vivo* angiogenesis assay and showed greater than 80% inhibition of corneal

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neovascularization (FIG. 5). While another antibody (10G4 19E6) was also identified as a potent inhibitor of VE-cadherin-mediated adherens junction formation by the in vitro assay criteria, this antibody disrupts existing junctions (FIG. 3). The key biological activities of these two antibodies are summarized in Table 3 along with data from other murine and human anti-VE-cadherin antibodies.